

STIC-ILL

QH301.F4

WCS

From: Prouty, Rebecca
Sent: Monday, February 11, 2002 2:18 PM
To: STIC-ILL
Subject: ILL Request

382738

Art Unit 1652
10A13, 308-4000
Mailbox: 10C01
Serial Number: 09/538,248

Please provide the following reference(s):

L169 ANSWER 7 OF 53 MEDLINE DUPLICATE 7

TI c-Src mediates mitogenic signals and associates with
cytoskeletal proteins upon vascular endothelial growth
factor stimulation in Kaposi's sarcoma cells.

SO JOURNAL OF IMMUNOLOGY, (2000 Feb 1) 164 (3) 1169-74.

Journal code: IFB; 2985117R. ISSN: 0022-1767.

AU Munshi N; Groopman J E; Gill P S; Ganju R K

AN 2000109065 MEDLINE

QH301.F33

L169 ANSWER 11 OF 53 SCISEARCH COPYRIGHT 2002 ISI (R) DUPLICATE 10

TI SRC-family kinase antagonist (PP2) inhibits VEGF-stimulated
VE-cadherin tyrosine phosphorylation in microvascular endothelial cells.

SO FASEB JOURNAL, (15 MAR 2000) Vol. 14, No. 4, pp. A145-A145.

Publisher: FEDERATION AMER SOC EXP BIOL, 9650 ROCKVILLE PIKE, BETHESDA, MD
20814-3998. ISSN: 0892-6638.

AU Cooke L S (Reprint); Forough R; Dawson N; Parrish A; Hoffman P; Kilgannon P; Granger H G

AN 2000:328497 SCISEARCH

L169 ANSWER 15 OF 53 MEDLINE DUPLICATE 12

TI Vascular endothelial growth factor signals endothelial cell production of
nitric oxide and prostacyclin through flk-1/KDR activation of c-Src.

SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1999 Aug 27) 274 (35) 25130-5.

Journal code: HIV; 2985121R. ISSN: 0021-9258.

AU He H; Venema V J; Gu X; Venema R C; Marrero M B; Caldwell R B

AN 1999387002 MEDLINE

L169 ANSWER 30 OF 53 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V. DUPLICATE 21

TI Tyrosine kinases in disease: Overview of kinase inhibitors as therapeutic
agents and current drugs in clinical trials.

SO Expert Opinion on Investigational Drugs, (1998) 7/4 (553-573).

Refs: 190 ISSN: 1354-3784 CODEN: EOIDER

AU Strawn L.M.; Shawver L.K.

AN 1998120483 EMBASE

L169 ANSWER 52 OF 53 MEDLINE DUPLICATE 35

TI Different signal transduction properties of KDR and Flt1, two receptors
for vascular endothelial growth factor.

SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1994 Oct 28) 269 (43) 26988-95.

Journal code: HIV; 2985121R. ISSN: 0021-9258.

AU Waltenberger J; Claesson-Welsh L; Siegbahn A; Shibuya M; Heldin C H

AN 95014567 MEDLINE

WR - NO (wrong
page
ordered)
CAS 2/14

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Journal code: HIV; 2985121R. ISSN: 0021-9258.

AU Waltenberger J; Claesson-Welsh L; Siegbahn A; Shibuya M; Heldin C H

AN 95014567 MEDLINE

* * * * * STN Columbus * * * * *

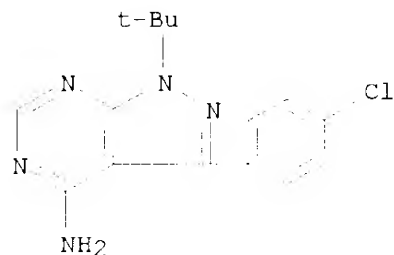
=> fil reg

=> s 172889-26-8 or 172889-26-9
1 172889-26-8
(172889-26-8/RN)
0 172889-26-9
(172889-26-9/RN)
L2 1 172889-26-8 OR 172889-26-9

=> s 172889-26-8 or 172889-27-9
1 172889-26-8
(172889-26-8/RN)
1 172889-27-9
(172889-27-9/RN)
L3 2 172889-26-8 OR 172889-27-9

=> d tot

L3 ANSWER 1 OF 2 REGISTRY COPYRIGHT 2002 ACS
RN **172889-27-9** REGISTRY
CN 1H-Pyrazolo[3,4-d]pyrimidin-4-amine, 3-(4-chlorophenyl)-1-(1,1-dimethylethyl)- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN PP 2
FS 3D CONCORD
MF C15 H16 Cl N5
SR CA
LC STN Files: CA, CAPLUS, CHEMCATS, TOXCENTER, TOXLIT, USPATFULL

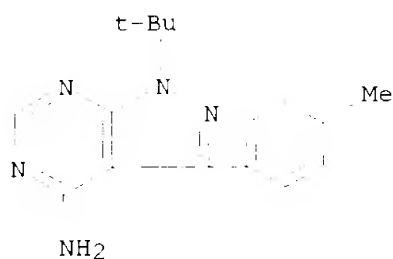


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

10 REFERENCES IN FILE CA (1967 TO DATE)
2 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
10 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L3 ANSWER 2 OF 2 REGISTRY COPYRIGHT 2002 ACS
RN **172889-26-8** REGISTRY
CN 1H-Pyrazolo[3,4-d]pyrimidin-4-amine, 1-(1,1-dimethylethyl)-3-(4-methylphenyl)- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN PP 1
FS 3D CONCORD
MF C16 H19 N5

SR CA
LC STN Files: CA, CAPLUS, CHEMCATS, TOXCENTER, TOXLIT, USPATFULL



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

20 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
20 REFERENCES IN FILE CAPLUS (1967 TO DATE)

=> log y

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
4.76	35.36

FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE	TOTAL
ENTRY	SESSION
0.00	-0.62

CA SUBSCRIBER PRICE

=> fil .bec

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
0.15	0.15

FULL ESTIMATED COST

FILES 'MEDLINE, SCISEARCH, LIFESCI, BIOTECHDS, BIOSIS, EMBASE, HCAPLUS, NTIS, ESBIODASE, BIOTECHNO, WPIDS' ENTERED AT 11:48:22 ON 11 FEB 2002
ALL COPYRIGHTS AND RESTRICTIONS APPLY. SEE HELP USAGETERMS FOR DETAILS.

11 FILES IN THE FILE LIST

=> s vascula?(3a)(permeab? or leak?)

FILE 'MEDLINE'

298739 VASCULA?

86873 PERMEAB?

31878 LEAK?

L1 10344 VASCULA?(3A)(PERMEAB? OR LEAK?)

FILE 'SCISEARCH'

174301 VASCULA?

80092 PERMEAB?

38371 LEAK?

L2 6521 VASCULA?(3A)(PERMEAB? OR LEAK?)

FILE 'LIFESCI'

20055 VASCULA?

15152 PERMEAB?
4677 LEAK?
L3 1032 VASCULA?(3A) (PERMEAB? OR LEAK?)

FILE 'BIOTECHDS'

1477 VASCULA?
2429 PERMEAB?
751 LEAK?
L4 46 VASCULA?(3A) (PERMEAB? OR LEAK?)

FILE 'BIOSIS'

812639 VASCULA?
81574 PERMEAB?
29396 LEAK?
L5 8035 VASCULA?(3A) (PERMEAB? OR LEAK?)

FILE 'EMBASE'

315413 VASCULA?
69207 PERMEAB?
29788 LEAK?
L6 5851 VASCULA?(3A) (PERMEAB? OR LEAK?)

FILE 'HCAPLUS'

109683 VASCULA?
174309 PERMEAB?
65622 LEAK?
L7 5809 VASCULA?(3A) (PERMEAB? OR LEAK?)

FILE 'NTIS'

2276 VASCULA?
12462 PERMEAB?
14526 LEAK?
L8 79 VASCULA?(3A) (PERMEAB? OR LEAK?)

FILE 'ESBIOBASE'

45773 VASCULA?
38894 PERMEAB?
6664 LEAK?
L9 1555 VASCULA?(3A) (PERMEAB? OR LEAK?)

FILE 'BIOTECHNO'

26349 VASCULA?
15091 PERMEAB?
3867 LEAK?
L10 1053 VASCULA?(3A) (PERMEAB? OR LEAK?)

FILE 'WPIDS'

13282 VASCULA?
89571 PERMEAB?
107887 LEAK?
L11 300 VASCULA?(3A) (PERMEAB? OR LEAK?)

TOTAL FOR ALL FILES

L12 40625 VASCULA?(3A) (PERMEAB? OR LEAK?)

=> s l12(8a) (inhibit? or decreas?)

FILE 'MEDLINE'

948649 INHIBIT?
760115 DECREAS?

L13 663 L1 (8A) (INHIBIT? OR DECREAS?)

FILE 'SCISEARCH'

732043 INHIBIT?

691479 DECREAS?

L14 448 L2 (8A) (INHIBIT? OR DECREAS?)

FILE 'LIFESCI'

262888 INHIBIT?

182622 DECREAS?

L15 90 L3 (8A) (INHIBIT? OR DECREAS?)

FILE 'BIOTECHDS'

33375 INHIBIT?

15496 DECREAS?

L16 6 L4 (8A) (INHIBIT? OR DECREAS?)

FILE 'BIOSIS'

1022135 INHIBIT?

885276 DECREAS?

L17 727 L5 (8A) (INHIBIT? OR DECREAS?)

FILE 'EMBASE'

843314 INHIBIT?

732340 DECREAS?

L18 672 L6 (8A) (INHIBIT? OR DECREAS?)

FILE 'HCAPLUS'

1461279 INHIBIT?

1871874 DECREAS?

L19 880 L7 (8A) (INHIBIT? OR DECREAS?)

FILE 'NTIS'

19267 INHIBIT?

49407 DECREAS?

L20 4 L8 (8A) (INHIBIT? OR DECREAS?)

FILE 'ESBIOBASE'

275631 INHIBIT?

217812 DECREAS?

L21 172 L9 (8A) (INHIBIT? OR DECREAS?)

FILE 'BIOTECHNO'

252731 INHIBIT?

144633 DECREAS?

L22 76 L10 (8A) (INHIBIT? OR DECREAS?)

FILE 'WPIDS'

172552 INHIBIT?

163615 DECREAS?

L23 57 L11 (8A) (INHIBIT? OR DECREAS?)

TOTAL FOR ALL FILES

L24 3795 L12 (8A) (INHIBIT? OR DECREAS?)

=> s 124 and (pyrazolopyrimidine# or ppl or pp2 or src or yes or 172889-26-8 or 172889-27-9)

FILE 'MEDLINE'

64 PYRAZOLOPYRIMIDINE#

973 PP1
 163 PP2
 10746 SRC
 2769 YES
 0 172889-26-8
 0 172889-27-9
 L25 1 L13 AND (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR
 172889-26-8 OR 172889-27-9)

FILE 'SCISEARCH'

99 PYRAZOLOPYRIMIDINE#
 979 PP1
 182 PP2
 10238 SRC
 2485 YES
 0 172889-26-8
 0 172889-27-9
 L26 1 L14 AND (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR
 172889-26-8 OR 172889-27-9)

FILE 'LIFESCI'

27 PYRAZOLOPYRIMIDINE#
 360 PP1
 43 PP2
 4432 SRC
 373 YES
 0 172889-26-8
 0 172889-27-9
 L27 0 L15 AND (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR
 172889-26-8 OR 172889-27-9)

FILE 'BIOTECHDS'

2 PYRAZOLOPYRIMIDINE#
 27 PP1
 11 PP2
 135 SRC
 24 YES
 0 172889-26-8
 0 172889-27-9
 L28 0 L16 AND (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR
 172889-26-8 OR 172889-27-9)

FILE 'BIOSIS'

115 PYRAZOLOPYRIMIDINE#
 1104 PP1
 228 PP2
 10426 SRC
 1520 YES
 0 172889-26-8
 0 172889-27-9
 L29 2 L17 AND (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR
 172889-26-8 OR 172889-27-9)

FILE 'EMBASE'

122 PYRAZOLOPYRIMIDINE#
 893 PP1
 123 PP2
 7957 SRC
 1922 YES

0 172889-26-8
0 172889-27-9
L30 1 L18 AND (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR
172889-26-8 OR 172889-27-9)

FILE 'HCAPLUS'

1137 PYRAZOLOPYRIMIDINE#
1187 PP1
274 PP2
10766 SRC
1478 YES
20 172889-26-8
10 172889-27-9
L31 2 L19 AND (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR
172889-26-8 OR 172889-27-9)

FILE 'NTIS'

1 PYRAZOLOPYRIMIDINE#
15 PP1
2 PP2
1937 SRC
307 YES
0 172889-26-8
0 172889-27-9
L32 0 L20 AND (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR
172889-26-8 OR 172889-27-9)

FILE 'ESBIOBASE'

9 PYRAZOLOPYRIMIDINE#
694 PP1
109 PP2
4886 SRC
463 YES
0 172889
44754 26
237842 8
0 172889-26-8
(172889(W)26(W)8)
0 172889
42591 27
177442 9
0 172889-27-9
(172889(W)27(W)9)
L33 1 L21 AND (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR
172889-26-8 OR 172889-27-9)

FILE 'BIOTECHNO'

15 PYRAZOLOPYRIMIDINE#
571 PP1
76 PP2
5674 SRC
307 YES
0 172889-26-8
0 172889-27-9
L34 1 L22 AND (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR
172889-26-8 OR 172889-27-9)

FILE 'WPIDS'

71 PYRAZOLOPYRIMIDINE#

140 PP1
 69 PP2
 462 SRC
 676 YES
 0 172889
 300469 26
 1687338 8
 0 172889-26-8
 (172889(W) 26(W) 8)
 0 172889
 186349 27
 1310082 9
 0 172889-27-9
 (172889(W) 27(W) 9)
 L35 3 L23 AND (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR
 172889-26-8 OR 172889-27-9)

TOTAL FOR ALL FILES

L36 12 L24 AND (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR
 172889-26-8 OR 172889-27-9)

=> s vascular endothelial or vpf or vegf

FILE 'MEDLINE'

279253 VASCULAR
 78248 ENDOTHELIAL
 10945 VASCULAR ENDOTHELIAL
 (VASCULAR(W) ENDOTHELIAL)
 278 VPF
 4522 VEGF
 L37 11478 VASCULAR ENDOTHELIAL OR VPF OR VEGF

FILE 'SCISEARCH'

160520 VASCULAR
 101259 ENDOTHELIAL
 13251 VASCULAR ENDOTHELIAL
 (VASCULAR(W) ENDOTHELIAL)
 387 VPF
 6026 VEGF
 L38 14872 VASCULAR ENDOTHELIAL OR VPF OR VEGF

FILE 'LIFESCI'

18149 "VASCULAR"
 13662 "ENDOTHELIAL"
 2261 VASCULAR ENDOTHELIAL
 ("VASCULAR"(W) "ENDOTHELIAL")
 70 VPF
 918 VEGF
 L39 2314 VASCULAR ENDOTHELIAL OR VPF OR VEGF

FILE 'BIOTECHDS'

1340 VASCULAR
 1266 ENDOTHELIAL
 364 VASCULAR ENDOTHELIAL
 (VASCULAR(W) ENDOTHELIAL)
 11 VPF
 159 VEGF
 L40 377 VASCULAR ENDOTHELIAL OR VPF OR VEGF

FILE 'BIOSIS'

793732 VASCULAR
101898 ENDOTHELIAL
16300 VASCULAR ENDOTHELIAL
 (VASCULAR(W) ENDOTHELIAL)
 342 VPF
 6383 VEGF
L41 16826 VASCULAR ENDOTHELIAL OR VPF OR VEGF

FILE 'EMBASE'
 294094 "VASCULAR"
 74125 "ENDOTHELIAL"
 10117 VASCULAR ENDOTHELIAL
 ("VASCULAR"(W) "ENDOTHELIAL")
 269 VPF
 4223 VEGF
L42 10689 VASCULAR ENDOTHELIAL OR VPF OR VEGF

FILE 'HCAPLUS'
 102821 VASCULAR
 61437 ENDOTHELIAL
 12482 VASCULAR ENDOTHELIAL
 (VASCULAR(W) ENDOTHELIAL)
 311 VPF
 4984 VEGF
L43 12861 VASCULAR ENDOTHELIAL OR VPF OR VEGF

FILE 'NTIS'
 2097 VASCULAR
 579 ENDOTHELIAL
 54 VASCULAR ENDOTHELIAL
 (VASCULAR(W) ENDOTHELIAL)
 36 VPF
 25 VEGF
L44 101 VASCULAR ENDOTHELIAL OR VPF OR VEGF

FILE 'ESBIOBASE'
 42688 VASCULAR
 36302 ENDOTHELIAL
 5684 VASCULAR ENDOTHELIAL
 (VASCULAR(W) ENDOTHELIAL)
 161 VPF
 3094 VEGF
L45 6043 VASCULAR ENDOTHELIAL OR VPF OR VEGF

FILE 'BIOTECHNO'
 24329 VASCULAR
 21443 ENDOTHELIAL
 4615 VASCULAR ENDOTHELIAL
 (VASCULAR(W) ENDOTHELIAL)
 157 VPF
 2498 VEGF
L46 4902 VASCULAR ENDOTHELIAL OR VPF OR VEGF

FILE 'WPIDS'
 12176 VASCULAR
 2932 ENDOTHELIAL
 831 VASCULAR ENDOTHELIAL
 (VASCULAR(W) ENDOTHELIAL)
 50 VPF

533 VEGF
L47 980 VASCULAR ENDOTHELIAL OR VPF OR VEGF

TOTAL FOR ALL FILES

L48 81443 VASCULAR ENDOTHELIAL OR VPF OR VEGF

=> s (pyrazolopyrimidine# or pp1 or pp2 or src or yes or 172889-26-8 or 172889-27-9)
FILE 'MEDLINE'

64 PYRAZOLOPYRIMIDINE#
973 PP1
163 PP2
10746 SRC
2769 YES
0 172889-26-8
0 172889-27-9
L49 14100 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR 172889-26-8
OR 172889-27-9)

FILE 'SCISEARCH'

99 PYRAZOLOPYRIMIDINE#
979 PP1
182 PP2
10238 SRC
2485 YES
0 172889-26-8
0 172889-27-9
L50 13461 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR 172889-26-8
OR 172889-27-9)

FILE 'LIFESCI'

27 PYRAZOLOPYRIMIDINE#
360 PP1
43 PP2
4432 SRC
373 YES
0 172889-26-8
0 172889-27-9
L51 5003 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR 172889-26-8
OR 172889-27-9)

FILE 'BIOTECHDS'

2 PYRAZOLOPYRIMIDINE#
27 PP1
11 PP2
135 SRC
24 YES
0 172889-26-8
0 172889-27-9
L52 188 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR 172889-26-8
OR 172889-27-9)

FILE 'BIOSIS'

115 PYRAZOLOPYRIMIDINE#
1104 PP1
228 PP2
10426 SRC
1520 YES
0 172889-26-8
0 172889-27-9

L53 12726 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR 172889-26-8
OR 172889-27-9)

FILE 'EMBASE'

122 PYRAZOLOPYRIMIDINE#
893 PP1
123 PP2
7957 SRC
1922 YES
0 172889-26-8
0 172889-27-9

L54 10540 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR 172889-26-8
OR 172889-27-9)

FILE 'HCAPLUS'

1137 PYRAZOLOPYRIMIDINE#
1187 PP1
274 PP2
10766 SRC
1478 YES
20 172889-26-8
10 172889-27-9

L55 14195 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR 172889-26-8
OR 172889-27-9)

FILE 'NTIS'

1 PYRAZOLOPYRIMIDINE#
15 PP1
2 PP2
1937 SRC
307 YES
0 172889-26-8
0 172889-27-9

L56 2260 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR 172889-26-8
OR 172889-27-9)

FILE 'ESBIOBASE'

9 PYRAZOLOPYRIMIDINE#
694 PP1
109 PP2
4886 SRC
463 YES
0 172889
44754 26
237842 8
0 172889-26-8
(172889(W) 26(W) 8)
0 172889
42591 27
177442 9
0 172889-27-9
(172889(W) 27(W) 9)

L57 5828 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR 172889-26-8
OR 172889-27-9)

FILE 'BIOTECHNO'

15 PYRAZOLOPYRIMIDINE#
571 PP1
76 PP2

5674 SRC
307 YES
0 172889-26-8
0 172889-27-9
L58 6329 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR 172889-26-8
OR 172889-27-9)

FILE 'WPIDS'

71 PYRAZOLOPYRIMIDINE#
140 PP1
69 PP2
462 SRC
676 YES
0 172889
300469 26
1687338 8
0 172889-26-8
(172889(W) 26(W) 8)
0 172889
186349 27
1310082 9
0 172889-27-9
(172889(W) 27(W) 9)
L59 1322 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR 172889-26-8
OR 172889-27-9)

TOTAL FOR ALL FILES

L60 85952 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR SRC OR YES OR 172889-26-8
OR 172889-27-9)

=> s 148 and 160

FILE 'MEDLINE'

L61 90 L37 AND L49

FILE 'SCISEARCH'

L62 96 L38 AND L50

FILE 'LIFESCI'

L63 21 L39 AND L51

FILE 'BIOTECHDS'

L64 0 L40 AND L52

FILE 'BIOSIS'

L65 97 L41 AND L53

FILE 'EMBASE'

L66 68 L42 AND L54

FILE 'HCAPLUS'

L67 109 L43 AND L55

FILE 'NTIS'

L68 0 L44 AND L56

FILE 'ESBIOBASE'

L69 56 L45 AND L57

FILE 'BIOTECHNO'

L70 43 L46 AND L58

FILE 'WPIDS'

L71 16 L47 AND L59

TOTAL FOR ALL FILES

L72 596 L48 AND L60

=> s 148(15a)160

FILE 'MEDLINE'

L73 40 L37(15A)L49

FILE 'SCISEARCH'

L74 44 L38(15A)L50

FILE 'LIFESCI'

L75 17 L39(15A)L51

FILE 'BIOTECHDS'

L76 0 L40(15A)L52

FILE 'BIOSIS'

L77 44 L41(15A)L53

FILE 'EMBASE'

L78 39 L42(15A)L54

FILE 'HCAPLUS'

L79 43 L43(15A)L55

FILE 'NTIS'

L80 0 L44(15A)L56

FILE 'ESBIOBASE'

L81 32 L45(15A)L57

FILE 'BIOTECHNO'

L82 27 L46(15A)L58

FILE 'WPIDS'

L83 6 L47(15A)L59

TOTAL FOR ALL FILES

L84 292 L48(15A) L60

=> s (src or yes)(8a)(inhibit? or decreas?)

FILE 'MEDLINE'

10746 SRC

2769 YES

948649 INHIBIT?

760115 DECREAS?

L85 1260 (SRC OR YES)(8A)(INHIBIT? OR DECREAS?)

FILE 'SCISEARCH'

10238 SRC

2485 YES

732043 INHIBIT?

691479 DECREAS?

L86 1225 (SRC OR YES) (8A) (INHIBIT? OR DECREAS?)

FILE 'LIFESCI'

4432 SRC
373 YES
262888 INHIBIT?
182622 DECREAS?

L87 494 (SRC OR YES) (8A) (INHIBIT? OR DECREAS?)

FILE 'BIOTECHDS'

135 SRC
24 YES
33375 INHIBIT?
15496 DECREAS?

L88 13 (SRC OR YES) (8A) (INHIBIT? OR DECREAS?)

FILE 'BIOSIS'

10426 SRC
1520 YES
1022135 INHIBIT?
885276 DECREAS?

L89 1408 (SRC OR YES) (8A) (INHIBIT? OR DECREAS?)

FILE 'EMBASE'

7957 SRC
1922 YES
843314 INHIBIT?
732340 DECREAS?

L90 1162 (SRC OR YES) (8A) (INHIBIT? OR DECREAS?)

FILE 'HCAPLUS'

10766 SRC
1478 YES
1461279 INHIBIT?
1871874 DECREAS?

L91 1416 (SRC OR YES) (8A) (INHIBIT? OR DECREAS?)

FILE 'NTIS'

1937 SRC
307 YES
19267 INHIBIT?
49407 DECREAS?

L92 22 (SRC OR YES) (8A) (INHIBIT? OR DECREAS?)

FILE 'ESBIOBASE'

4886 SRC
463 YES
275631 INHIBIT?
217812 DECREAS?

L93 821 (SRC OR YES) (8A) (INHIBIT? OR DECREAS?)

FILE 'BIOTECHNO'

5674 SRC
307 YES
252731 INHIBIT?
144633 DECREAS?

L94 729 (SRC OR YES) (8A) (INHIBIT? OR DECREAS?)

FILE 'WPIDS'

```

        462 SRC
        676 YES
        172552 INHIBIT?
        163615 DECREAS?
L95      80 (SRC OR YES) (8A) (INHIBIT? OR DECREAS?)

TOTAL FOR ALL FILES
L96      8630 (SRC OR YES) (8A) (INHIBIT? OR DECREAS?)

=> s (pyrazolopyrimidine# or pp1 or pp2 or 172889-26-8 or 172889-27-9)
FILE 'MEDLINE'
        64 PYRAZOLOPYRIMIDINE#
        973 PP1
        163 PP2
            0 172889-26-8
            0 172889-27-9
L97      1153 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR 172889-26-8 OR 172889-27-9
            )

FILE 'SCISEARCH'
        99 PYRAZOLOPYRIMIDINE#
        979 PP1
        182 PP2
            0 172889-26-8
            0 172889-27-9
L98      1216 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR 172889-26-8 OR 172889-27-9
            )

FILE 'LIFESCI'
        27 PYRAZOLOPYRIMIDINE#
        360 PP1
        43 PP2
            0 172889-26-8
            0 172889-27-9
L99      416 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR 172889-26-8 OR 172889-27-9
            )

FILE 'BIOTECHDS'
        2 PYRAZOLOPYRIMIDINE#
        27 PP1
        11 PP2
            0 172889-26-8
            0 172889-27-9
L100     35 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR 172889-26-8 OR 172889-27-9
            )

FILE 'BIOSIS'
        115 PYRAZOLOPYRIMIDINE#
        1104 PP1
        228 PP2
            0 172889-26-8
            0 172889-27-9
L101     1385 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR 172889-26-8 OR 172889-27-9
            )

FILE 'EMBASE'
        122 PYRAZOLOPYRIMIDINE#
        893 PP1
        123 PP2

```


0 172889-26-8
0 172889-27-9
L102 1105 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR 172889-26-8 OR 172889-27-9
)

FILE 'HCAPLUS'

1137 PYRAZOLOPYRIMIDINE#
1187 PP1
274 PP2
20 172889-26-8
10 172889-27-9
L103 2532 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR 172889-26-8 OR 172889-27-9
)

FILE 'NTIS'

1 PYRAZOLOPYRIMIDINE#
15 PP1
2 PP2
0 172889-26-8
0 172889-27-9
L104 18 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR 172889-26-8 OR 172889-27-9
)

FILE 'ESBIOBASE'

9 PYRAZOLOPYRIMIDINE#
694 PP1
109 PP2
0 172889
44754 26
237842 8
0 172889-26-8
(172889(W) 26(W) 8)
0 172889
42591 27
177442 9
0 172889-27-9
(172889(W) 27(W) 9)
L105 784 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR 172889-26-8 OR 172889-27-9
)

FILE 'BIOTECHNO'

15 PYRAZOLOPYRIMIDINE#
571 PP1
76 PP2
0 172889-26-8
0 172889-27-9
L106 645 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR 172889-26-8 OR 172889-27-9
)

FILE 'WPIDS'

71 PYRAZOLOPYRIMIDINE#
140 PP1
69 PP2
0 172889
300469 26
1687338 8
0 172889-26-8
(172889(W) 26(W) 8)
0 172889

186349 27
1310082 9
0 172889-27-9
(172889(W)27(W)9)
L107 225 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR 172889-26-8 OR 172889-27-9
)

TOTAL FOR ALL FILES

L108 9514 (PYRAZOLOPYRIMIDINE# OR PP1 OR PP2 OR 172889-26-8 OR 172889-27-9
)

=> s 196 and 1108

FILE 'MEDLINE'

L109 252 L85 AND L97

FILE 'SCISEARCH'

L110 234 L86 AND L98

FILE 'LIFESCI'

L111 83 L87 AND L99

FILE 'BIOTECHDS'

L112 0 L88 AND L100

FILE 'BIOSIS'

L113 307 L89 AND L101

FILE 'EMBASE'

L114 217 L90 AND L102

FILE 'HCAPLUS'

L115 253 L91 AND L103

FILE 'NTIS'

L116 1 L92 AND L104

FILE 'ESBIOBASE'

L117 195 L93 AND L105

FILE 'BIOTECHNO'

L118 135 L94 AND L106

FILE 'WPIDS'

L119 3 L95 AND L107

TOTAL FOR ALL FILES

L120 1680 L96 AND L108

=> s 196(5a)1108

FILE 'MEDLINE'

L121 236 L85(5A)L97

FILE 'SCISEARCH'

L122 213 L86(5A)L98

FILE 'LIFESCI'

L123 78 L87(5A)L99

FILE 'BIOTECHDS'

L124 0 L88(5A)L100
 FILE 'BIOSIS'
 L125 284 L89(5A)L101
 FILE 'EMBASE'
 L126 194 L90(5A)L102
 FILE 'HCAPLUS'
 L127 231 L91(5A)L103
 FILE 'NTIS'
 L128 1 L92(5A)L104
 FILE 'ESBIOBASE'
 L129 179 L93(5A)L105
 FILE 'BIOTECHNO'
 L130 128 L94(5A)L106
 FILE 'WPIDS'
 L131 1 L95(5A)L107
 TOTAL FOR ALL FILES
 L132 1545 L96(5A) L108
 => s l24 and l108
 FILE 'MEDLINE'
 L133 0 L13 AND L97
 FILE 'SCISEARCH'
 L134 0 L14 AND L98
 FILE 'LIFESCI'
 L135 0 L15 AND L99
 FILE 'BIOTECHDS'
 L136 0 L16 AND L100
 FILE 'BIOSIS'
 L137 1 L17 AND L101
 FILE 'EMBASE'
 L138 0 L18 AND L102
 FILE 'HCAPLUS'
 L139 1 L19 AND L103
 FILE 'NTIS'
 L140 0 L20 AND L104
 FILE 'ESBIOBASE'
 L141 0 L21 AND L105
 FILE 'BIOTECHNO'
 L142 0 L22 AND L106
 FILE 'WPIDS'
 L143 0 L23 AND L107

TOTAL FOR ALL FILES

L144 2 L24 AND L108

=> s 148 and 1108

FILE 'MEDLINE'

L145 10 L37 AND L97

FILE 'SCISEARCH'

L146 14 L38 AND L98

FILE 'LIFESCI'

L147 2 L39 AND L99

FILE 'BIOTECHDS'

L148 0 L40 AND L100

FILE 'BIOSIS'

L149 14 L41 AND L101

FILE 'EMBASE'

L150 8 L42 AND L102

FILE 'HCAPLUS'

L151 15 L43 AND L103

FILE 'NTIS'

L152 0 L44 AND L104

FILE 'ESBIOBASE'

L153 8 L45 AND L105

FILE 'BIOTECHNO'

L154 4 L46 AND L106

FILE 'WPIDS'

L155 1 L47 AND L107

TOTAL FOR ALL FILES

L156 76 L48 AND L108

=> s (136 or 184 or 1144 or 1156) not 2001-2002/py

FILE 'MEDLINE'

501323 2001-2002/PY

L157 33 (L25 OR L73 OR L133 OR L145) NOT 2001-2002/PY

FILE 'SCISEARCH'

969262 2001-2002/PY

L158 39 (L26 OR L74 OR L134 OR L146) NOT 2001-2002/PY

FILE 'LIFESCI'

64358 2001-2002/PY

L159 15 (L27 OR L75 OR L135 OR L147) NOT 2001-2002/PY

FILE 'BIOTECHDS'

11462 2001-2002/PY

L160 0 (L28 OR L76 OR L136 OR L148) NOT 2001-2002/PY

FILE 'BIOSIS'

461214 2001-2002/PY
L161 38 (L29 OR L77 OR L137 OR L149) NOT 2001-2002/PY

FILE 'EMBASE'

433129 2001-2002/PY
L162 35 (L30 OR L78 OR L138 OR L150) NOT 2001-2002/PY

FILE 'HCAPLUS'

1027851 2001-2002/PY
L163 32 (L31 OR L79 OR L139 OR L151) NOT 2001-2002/PY

FILE 'NTIS'

0 2001-2002/PY
L164 0 (L32 OR L80 OR L140 OR L152) NOT 2001-2002/PY

FILE 'ESBIOBASE'

271702 2001-2002/PY
L165 27 (L33 OR L81 OR L141 OR L153) NOT 2001-2002/PY

FILE 'BIOTECHNO'

114906 2001-2002/PY
L166 25 (L34 OR L82 OR L142 OR L154) NOT 2001-2002/PY

FILE 'WPIDS'

889018 2001-2002/PY
L167 0 (L35 OR L83 OR L143 OR L155) NOT 2001-2002/PY

TOTAL FOR ALL FILES

L168 244 (L36 OR L84 OR L144 OR L156) NOT 2001-2002/PY

=> dup rem l168

PROCESSING COMPLETED FOR L168

L169 53 DUP REM L168 (191 DUPLICATES REMOVED)

=> d tot

L169 ANSWER 1 OF 53 MEDLINE DUPLICATE 1
TI Collagen, convulxin, and thrombin stimulate aggregation-independent tyrosine phosphorylation of CD31 in platelets. Evidence for the involvement of Src family kinases.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (2000 Sep 1) 275 (35) 27339-47.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
AU Cicmil M; Thomas J M; Sage T; Barry F A; Leduc M; Bon C; Gibbins J M
AN 2000472698 MEDLINE

L169 ANSWER 2 OF 53 MEDLINE DUPLICATE 2
TI Norepinephrine induces **vascular endothelial** growth factor gene expression in brown adipocytes through a beta -adrenoreceptor/cAMP/protein kinase A pathway involving Src but independently of Erk1/2.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (2000 May 5) 275 (18) 13802-11.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
AU Fredriksson J M; Lindquist J M; Bronnikov G E; Nedergaard J
AN 2000250944 MEDLINE

L169 ANSWER 3 OF 53 MEDLINE DUPLICATE 3
TI Oncogenes and tumor angiogenesis: the HPV-16 E6 oncoprotein activates the vascular endothelial growth factor (VEGF) gene promoter in a p53 independent manner.

SO ONCOGENE, (2000 Sep 21) 19 (40) 4611-20.
Journal code: ONC. ISSN: 0950-9232.

AU Lopez-Ocejo O; Vilorio-Petit A; Bequet-Romero M; Mukhopadhyay D; Rak J;
Kerbel R S

AN 2000478410 MEDLINE

L169 ANSWER 4 OF 53 MEDLINE DUPLICATE 4

TI Identification of substituted 3-[(4,5,6, 7-tetrahydro-1H-indol-2-yl)methylene]-1,3-dihydroindol-2-ones as growth factor receptor inhibitors for VEGF-R2 (Flk-1/KDR), FGF-R1, and PDGF-Rbeta tyrosine kinases.

SO JOURNAL OF MEDICINAL CHEMISTRY, (2000 Jul 13) 43 (14) 2655-63.
Journal code: JOF; 9716531. ISSN: 0022-2623.

AU Sun L; Tran N; Liang C; Hubbard S; Tang F; Lipson K; Schreck R; Zhou Y; McMahon G; Tang C

AN 2000353480 MEDLINE

L169 ANSWER 5 OF 53 MEDLINE DUPLICATE 5

TI PTK787/ZK 222584, a novel and potent inhibitor of vascular endothelial growth factor receptor tyrosine kinases, impairs vascular endothelial growth factor-induced responses and tumor growth after oral administration.

SO CANCER RESEARCH, (2000 Apr 15) 60 (8) 2178-89.
Journal code: CNF; 2984705R. ISSN: 0008-5472.

AU Wood J M; Bold G; Buchdunger E; Cozens R; Ferrari S; Frei J; Hofmann F; Mestan J; Mett H; O'Reilly T; Persohn E; Rosel J; Schnell C; Stover D; Theuer A; Towbin H; Wenger F; Woods-Cook K; Menrad A; Siemeister G; Schirner M; Thierauch K H; Schneider M R; Dreys J; Martiny-Baron G; Totzke F

AN 2000246354 MEDLINE

L169 ANSWER 6 OF 53 MEDLINE DUPLICATE 6

TI Opposite effects of pressurized steady versus pulsatile perfusion on **vascular endothelial** cell cytosolic pH: role of tyrosine kinase and mitogen-activated protein kinase signaling.

SO CIRCULATION RESEARCH, (2000 Jun 23) 86 (12) 1230-6.
Journal code: DAJ; 0047103. ISSN: 1524-4571.

AU Wittstein I S; Qiu W; Ziegelstein R C; Hu Q; Kass D A

AN 2000325061 MEDLINE

L169 ANSWER 7 OF 53 MEDLINE DUPLICATE 7

TI c-**Src** mediates mitogenic signals and associates with cytoskeletal proteins upon **vascular endothelial** growth factor stimulation in Kaposi's sarcoma cells.

SO JOURNAL OF IMMUNOLOGY, (2000 Feb 1) 164 (3) 1169-74.
Journal code: IFB; 2985117R. ISSN: 0022-1767.

AU Munshi N; Groopman J E; Gill P S; Ganju R K

AN 2000109065 MEDLINE

L169 ANSWER 8 OF 53 MEDLINE DUPLICATE 8

TI Induction by carbon-ion irradiation of the expression of vascular endothelial growth factor in lung carcinoma cells.

SO INTERNATIONAL JOURNAL OF RADIATION BIOLOGY, (2000 Aug) 76 (8) 1121-7.
Journal code: IRB; 8809243. ISSN: 0955-3002.

AU Ando S; Nojima K; Ishihara H; Suzuki M; Ando M; Majima H; Ando K; Kuriyama T

AN 2000413801 MEDLINE

L169 ANSWER 9 OF 53 MEDLINE DUPLICATE 9

TI The Shc-related adaptor protein, Sck, forms a complex with the

- vascular-endothelial-growth-factor receptor KDR in transfected cells.
 SO BIOCHEMICAL JOURNAL, (2000 Apr 15) 347 (Pt 2) 501-9.
 Journal code: 9YO; 2984726R. ISSN: 0264-6021.
 AU Warner A J; Lopez-Dee J; Knight E L; Feramisco J R; Prigent S A
 AN 2001132966 MEDLINE
- L169 ANSWER 10 OF 53 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
 TI Intercellular signaling within vascular cells under hyperglycemia involves
 free radical-triggered tyrosine kinase activation.
 SO Journal of Submicroscopic Cytology and Pathology, (July, 2000) Vol. 32,
 No. 3, pp. 471. print.
 Meeting Info.: XIth International Vascular Biology Meeting Geneva,
 Switzerland September 05-09, 2000
 ISSN: 1122-9497.
 AU Graier, W. F. (1); Schaeffer, G. (1); Levak-Frank, S. (1); Spitaler, M. M.
 (1)
 AN 2001:100521 BIOSIS
- L169 ANSWER 11 OF 53 SCISEARCH COPYRIGHT 2002 ISI (R) DUPLICATE 10
 TI **Src**-family kinase antagonist (**PP2**) inhibits
VEGF-stimulated VE-cadherin tyrosine phosphorylation in
 microvascular endothelial cells.
 SO FASEB JOURNAL, (15 MAR 2000) Vol. 14, No. 4, pp. A145-A145.
 Publisher: FEDERATION AMER SOC EXP BIOL, 9650 ROCKVILLE PIKE, BETHESDA, MD
 20814-3998.
 ISSN: 0892-6638.
 AU Cooke L S (Reprint); Forough R; Dawson N; Parrish A; Hoffman P; Kilgannon
 P; Granger H G
 AN 2000:328497 SCISEARCH
- L169 ANSWER 12 OF 53 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
 TI The mechanism by which angiopoietin 1 **inhibits VEGF**
-mediated vascular permeability.
 SO Blood, (November 16, 2000) Vol. 96, No. 11 Part 2, pp. 62b. print.
 Meeting Info.: 42nd Annual Meeting of the American Society of Hematology
 San Francisco, California, USA December 01-05, 2000 American Society of
 Hematology
 . ISSN: 0006-4971.
 AU Wang, Yihong (1); Pampou, Sergei; Varticovski, Lyuba (1)
 AN 2001:312197 BIOSIS
- L169 ANSWER 13 OF 53 MEDLINE DUPLICATE 11
 TI Inhibition of vascular endothelial growth factor (VEGF) as a novel
 approach for cancer therapy.
 SO MEDICINA, (2000) 60 Suppl 2 41-7.
 Journal code: MMM. ISSN: 0025-7680.
 AU Wood J M
 AN 2001098649 MEDLINE
- L169 ANSWER 14 OF 53 SCISEARCH COPYRIGHT 2002 ISI (R)
 TI Shear stress stimulation of p130(cas) tyrosine phosphorylation requires
 calcium-dependent c-Src activation
 SO JOURNAL OF BIOLOGICAL CHEMISTRY, (17 SEP 1999) Vol. 274, No. 38, pp.
 26803-26809.
 Publisher: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE
 PIKE, BETHESDA, MD 20814.
 ISSN: 0021-9258.
 AU Okuda M; Takahashi M; Suero J; Murry C E; Traub O; Kawakatsu H; Berk B C
 (Reprint)

AN 1999:719861 SCISEARCH

L169 ANSWER 15 OF 53 MEDLINE DUPLICATE 12
TI Vascular endothelial growth factor signals endothelial cell production of nitric oxide and prostacyclin through flk-1/KDR activation of c-Src.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1999 Aug 27) 274 (35) 25130-5.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
AU He H; Venema V J; Gu X; Venema R C; Marrero M B; Caldwell R B
AN 1999387002 MEDLINE

L169 ANSWER 16 OF 53 MEDLINE DUPLICATE 13
TI Decreased Src tyrosine kinase activity inhibits malignant human ovarian cancer tumor growth in a nude mouse model.
SO CLINICAL CANCER RESEARCH, (1999 Aug) 5 (8) 2164-70.
Journal code: C2H; 9502500. ISSN: 1078-0432.
AU Wiener J R; Nakano K; Kruzelock R P; Bucana C D; Bast R C Jr; Gallick G E
AN 1999400080 MEDLINE

L169 ANSWER 17 OF 53 MEDLINE DUPLICATE 14
TI Vascular endothelial growth factor induces activation and subcellular translocation of focal adhesion kinase (p125FAK) in cultured rat cardiac myocytes.
SO CIRCULATION RESEARCH, (1999 May 28) 84 (10) 1194-202.
Journal code: DAJ; 0047103. ISSN: 0009-7330.
AU Takahashi N; Seko Y; Noiri E; Tobe K; Kadowaki T; Sabe H; Yazaki Y
AN 1999278200 MEDLINE

L169 ANSWER 18 OF 53 SCISEARCH COPYRIGHT 2002 ISI (R)
TI 'Outside-in' signalling mechanisms underlying CD11b/CD18-mediated NADPH oxidase activation in human adherent blood eosinophils
SO BRITISH JOURNAL OF PHARMACOLOGY, (NOV 1999) Vol. 128, No. 6, pp. 1149-1158.
Publisher: STOCKTON PRESS, HOUNDMILLS, BASINGSTOKE RG21 6XS, HAMPSHIRE, ENGLAND.
ISSN: 0007-1188.
AU Lynch O T; Giembycz M A; Barnes P J; Hellewell P G; Lindsay M A (Reprint)
AN 1999:934514 SCISEARCH

L169 ANSWER 19 OF 53 MEDLINE DUPLICATE 15
TI Selective requirement for **Src** kinases during **VEGF**-induced angiogenesis and vascular permeability.
SO MOLECULAR CELL, (1999 Dec) 4 (6) 915-24.
Journal code: C5E; 9802571. ISSN: 1097-2765.
AU Eliceiri B P; Paul R; Schwartzberg P L; Hood J D; Leng J; Cheresch D A
AN 2000101234 MEDLINE

L169 ANSWER 20 OF 53 MEDLINE DUPLICATE 16
TI Early induction of angiogenetic signals in gliomas of GFAP-v-src transgenic mice.
SO AMERICAN JOURNAL OF PATHOLOGY, (1999 Feb) 154 (2) 581-90.
Journal code: 3RS; 0370502. ISSN: 0002-9440.
AU Theurillat J P; Hainfellner J; Maddalena A; Weissenberger J; Aguzzi A
AN 1999149661 MEDLINE

L169 ANSWER 21 OF 53 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI Role of tyrosine residues and protein interaction domains of SHC adaptor in VEGF receptor 3 signaling.
SO Oncogene, (Jan. 14, 1999) Vol. 18, No. 2, pp. 507-514.
ISSN: 0950-9232.

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Birnbaum, Daniel (1); Borg, Jean-Paul
AN 1999:124999 BIOSIS

L169 ANSWER 22 OF 53 HCAPLUS COPYRIGHT 2002 ACS
TI Protein phosphorylation in shear stress activated endothelial cells
SO Endothelial Cell Res. Ser. (1999), 6(Mechanical Forces and the
Endothelium), 69-87
CODEN: ECRSFY; ISSN: 1384-1270
AU Shyy, John Y.-J.; Li, Yi-Shuan; Li, Song; Jalali, Shila; Kim, Michael;
Usami, Shunichi; Chien, Shu
AN 1999:653131 HCAPLUS
DN 132:91115

L169 ANSWER 23 OF 53 MEDLINE DUPLICATE 17
TI A dual inhibitor of platelet-derived growth factor beta-receptor and Src
kinase activity potently interferes with mitogenic and mitogenic responses
to PDGF in vascular smooth muscle cells. A novel candidate for prevention
of vascular remodeling.
SO CIRCULATION RESEARCH, (1999 Jul 9) 85 (1) 12-22.
Journal code: DAJ; 0047103. ISSN: 1524-4571.
AU Waltenberger J; Uecker A; Kroll J; Frank H; Mayr U; Bjorge J D; Fujita D;
Gazit A; Hombach V; Levitzki A; Bohmer F D
AN 1999330572 MEDLINE

L169 ANSWER 24 OF 53 HCAPLUS COPYRIGHT 2002 ACS
TI Fusion proteins containing multiple domains binding to a target protein
and their investigative and therapeutic uses
SO PCT Int. Appl., 58 pp.
CODEN: PIXXD2
IN Cowburn, David; Zheng, Jie; Barany, George; Xu, Qinhong
AN 1998:251265 HCAPLUS
DN 128:305398
PATENT NO. KIND DATE APPLICATION NO. DATE

PI WO 9816638 A1 19980423 WO 1996-US16495 19961016
W: AU, CA, JP, MX
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
AU 9674324 A1 19980511 AU 1996-74324 19961016

L169 ANSWER 25 OF 53 MEDLINE DUPLICATE 18
TI Homologous up-regulation of KDR/Flk-1 receptor expression by vascular
endothelial growth factor in vitro.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1998 Nov 6) 273 (45) 29979-85.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
AU Shen B Q; Lee D Y; Gerber H P; Keyt B A; Ferrara N; Zioncheck T F
AN 1999009119 MEDLINE

L169 ANSWER 26 OF 53 MEDLINE DUPLICATE 19
TI Identification of vascular endothelial growth factor receptor-1 tyrosine
phosphorylation sites and binding of SH2 domain-containing molecules.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1998 Sep 4) 273 (36) 23410-8.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
AU Ito N; Wernstedt C; Engstrom U; Claesson-Welsh L
AN 1998389777 MEDLINE

L169 ANSWER 27 OF 53 LIFESCI COPYRIGHT 2002 CSA
TI Mitochondrial reactive oxygen species trigger hypoxia-induced
transcription

SO Proc. Natl. Acad. Sci. USA, (19980929) vol. 95, no. 20, pp. 11715-11720.
ISSN: 0027-8424.
AU Chandel, N.S.; Maltepe, E.; Goldwasser, E.; Mathieu, C.E.; Simon, M.C.;
Schumacker, P.T.
AN 1999:14022 LIFESCI

L169 ANSWER 28 OF 53 SCISEARCH COPYRIGHT 2002 ISI (R)
TI Requirement for **Src** activity during **VEGF** but not
bFGF-induced angiogenesis.
SO MOLECULAR BIOLOGY OF THE CELL, (NOV 1998) Vol. 9, Supp. [S], pp.
2444-2444.
Publisher: AMER SOC CELL BIOLOGY, PUBL OFFICE, 9650 ROCKVILLE PIKE,
BETHESDA, MD 20814.
ISSN: 1059-1524.
AU Eliceiri B P (Reprint); Andrews C; Schwartzerg P L; Cheresch D A
AN 1998:908359 SCISEARCH

L169 ANSWER 29 OF 53 MEDLINE DUPLICATE 20
TI Down-regulation of vascular endothelial growth factor in a human colon
carcinoma cell line transfected with an antisense expression vector
specific for c-src.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1998 Jan 9) 273 (2) 1052-7.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
AU Ellis L M; Staley C A; Liu W; Fleming R Y; Parikh N U; Bucana C D; Gallick
G E
AN 1998086282 MEDLINE

L169 ANSWER 30 OF 53 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 21
TI Tyrosine kinases in disease: Overview of kinase inhibitors as therapeutic
agents and current drugs in clinical trials.
SO Expert Opinion on Investigational Drugs, (1998) 7/4 (553-573).
Refs: 190
ISSN: 1354-3784 CODEN: EOIDER
AU Strawn L.M.; Shawver L.K.
AN 1998120483 EMBASE

L169 ANSWER 31 OF 53 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI Functional characterization of SRC kinases in Kaposi's Sarcoma (KS) cells.
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Meeting Info.: 40th Annual Meeting of the American Society of Hematology
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ANSWER SET L169 HAS BEEN SAVED AS 'PP1/A'

=> d ab 7,11,15,20,30,35,39,52

L121 ANSWER 7 OF 53 MEDLINE DUPLICATE 7
AB **Vascular endothelial** growth factor (**VEGF**)
appears to be a critical cytokine modulating the growth and spread of
Kaposi's sarcoma (KS). Furthermore, infection with the KS herpes virus
results in up-regulation of **VEGF** and triggering of **VEGF**
receptor activation. The molecular mechanisms regulating such
cytokine-driven proliferation of KS cells are not well characterized. We
investigated the role of **Src**-related tyrosine kinases in
VEGF-mediated signaling in model KS 38 tumor cells. **VEGF**
stimulation specifically activated c-**Src** kinase activity but not
that of other related **Src** kinases such as Lyn, Fyn, or Hck in KS
cells. **Pyrazolopyrimidine**, a selective inhibitor of **Src**
family tyrosine kinases, significantly blocked the **VEGF**-induced
growth of KS cells. Further studies using mutants of c-**Src**
kinase revealed that **Src** mediates mitogen-activated protein
kinase activation induced by **VEGF**. We also observed that
VEGF stimulation resulted in increased tyrosine phosphorylation of
the focal adhesion components paxillin and p130cas. Furthermore,
VEGF induction enhanced the complex formation between **Src**
kinase and paxillin. **Src** kinase appears to play an important
functional role in **VEGF**-induced signaling in KS cells and may
act to link pathways from the **VEGF** receptor to mitogen-activated
protein kinase and cytoskeletal components, thereby effecting tumor
proliferation and migration.

L121 ANSWER 11 OF 53 SCISEARCH COPYRIGHT 2002 ISI (R) DUPLICATE 10

L121 ANSWER 15 OF 53 MEDLINE DUPLICATE 12
AB Vascular endothelial growth factor (VEGF) is a potent endothelial
cell-specific mitogen that promotes angiogenesis, vascular
hyperpermeability, and vasodilation by autocrine mechanisms involving
nitric oxide (NO) and prostacyclin (PGI(2)) production. These experiments
used immunoprecipitation and immunoassay procedures to characterize the
signaling pathways by which VEGF induces NO and PGI(2) formation in
cultured endothelial cells. The data showed that VEGF stimulates complex
formation of the flk-1/kinase-insert domain-containing receptor (KDR)
VEGF receptor with c-**Src** and that **Src**
activation is required for **VEGF** induction of phospholipase C
gamma1 activation and inositol 1,4,5-trisphosphate formation. Reporter
cell assays showed that VEGF promotes a approximately 50-fold increase in

NO formation, which peaks at 5-20 min. This effect is mediated by a signaling cascade initiated by flk-1/KDR activation of c-Src, leading to phospholipase C gamma1 activation, inositol 1,4,5-trisphosphate formation, release of $[Ca^{2+}]_i$ and nitric oxide synthase activation. Immunoassays of VEGF-induced 6-keto prostaglandin F(1alpha) formation as an indicator of PGI(2) production revealed a 3-4-fold increase that peaked at 45-60 min. The PGI(2) signaling pathway follows the NO pathway through release of $[Ca^{2+}]_i$, but diverges prior to NOS activation and also requires activation of mitogen-activated protein kinase. These results suggest that NO and PGI(2) function in parallel in mediating the effects of VEGF.

L121 ANSWER 20 OF 53 MEDLINE DUPLICATE 16

AB Angiogenesis is a prerequisite for solid tumor growth. Glioblastoma multiforme, the most common malignant brain tumor, is characterized by extensive vascular proliferation. We previously showed that transgenic mice expressing a GFAP-v-src fusion gene in astrocytes develop low-grade astrocytomas that progressively evolve into hypervascularized glioblastomas. Here, we examined whether tumor progression triggers angiogenetic signals. We found abundant transcription of **vascular endothelial** growth factor (VEGF) in neoplastic astrocytes at surprisingly early stages of tumorigenesis. VEGF and v-src expression patterns were not identical, suggesting that VEGF activation was not only dependent on v-src. Late-stage gliomas showed perinecrotic VEGF up-regulation similarly to human glioblastoma. Expression patterns of the endothelial angiogenic receptors flt-1, flk-1, tie-1, and tie-2 were similar to those described in human gliomas, but flt-1 was expressed also in neoplastic astrocytes, suggesting an autocrine role in tumor growth. In crossbreeding experiments, hemizygous ablation of the tumor suppressor genes Rb and p53 had no significant effect on the expression of VEGF, flt-1, flk-1, tie-1, and tie-2. Therefore, expression of angiogenic signals is an early event during progression of GFAP-v-src tumors and precedes hypervascularization. Given the close similarities in the progression pattern between GFAP-v-src and human gliomas, the present results suggest that these mice may provide a useful tool for antiangiogenic therapy research.

L121 ANSWER 30 OF 53 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 21

AB Tyrosine kinases, first described as oncogenes, have been shown to play a role in normal cellular processes. Aberrations in tyrosine kinase activity lead to disease states. For fifteen years it has been postulated that the inhibition of tyrosine kinases may have therapeutic utility and the design and testing of inhibitors have been major focuses of research and development in both academic institutions and pharmaceutical companies. While early research focused on developing chemical entities that mimic phosphotyrosine, later research has focused on developing competitive adenosine triphosphate (ATP) inhibitors with various levels of selectivity on kinase targets. This review focuses on a discussion of tyrosine kinases thought to be important in disease, including platelet-derived growth factor (PDGF), fibroblast growth factor (FGF), **vascular endothelial** cell growth factor (VEGF), epidermal growth factor (EGF) receptors, HER-2 and Src. In addition, the classes of inhibitors designed to affect these targets and that have overcome research and development challenges and entered clinical trials are discussed. These include isoxazole, quinazoline, substituted pyrimidines and indolinone compounds, all of which are in clinical trials or near clinical development by SUGEN, Zeneca, Novartis, Pfizer and Parke-Davis. A summary of the chemistry and activity of these agents is provided.

L121 ANSWER 35 OF 53 MEDLINE DUPLICATE 24

AB Hemodynamic abnormalities have been implicated in the pathogenesis of the increased glomerular permeability to protein of diabetic and other glomerulopathies. Vascular permeability factor (VPF) is one of the most powerful promoters of vascular permeability. We studied the effect of stretch on VPF production by human mesangial cells and the intracellular signaling pathways involved. The application of mechanical stretch (elongation 10%) for 6 h induced a 2.4-fold increase over control in the VPF mRNA level ($P < 0.05$). There was a corresponding 3-fold increase in VPF protein level by 12 h ($P < 0.001$), returning to the baseline by 24 h. Stretch-induced VPF secretion was partially prevented both by the protein kinase C (PKC) inhibitor H7 (50 μ M: 72% inhibition, $P < 0.05$) and by pretreatment with phorbol ester (phorbol-12-myristate-13 acetate 10^{-7} M: 77% inhibition, $P < 0.05$). A variety of protein tyrosine kinase (PTK) inhibitors, genistein (20 μ g/ml), herbimycin A (3.4 μ M), and a specific pp60(**src**) peptide inhibitor (21 μ M) also significantly reduced, but did not entirely prevent, stretch-induced VPF protein secretion (respectively 63%, 80%, and 75% inhibition; $P < 0.05$ for all). The combination of both PKC and PTK inhibition completely abolished the VPF response to mechanical stretch (100% inhibition, $P < 0.05$). Stretch induces VPF gene expression and protein secretion in human mesangial cells via PKC- and PTK-dependent mechanisms.

L121 ANSWER 39 OF 53 SCISEARCH COPYRIGHT 2002 ISI (R) DUPLICATE 27

AB Balloon angioplasty disrupts the protective endothelial lining of the arterial wall, rendering arteries susceptible to thrombosis and intimal thickening. We show here that Vascular endothelial growth factor (VEGF), an endothelial cell mitogen, is upregulated in medial smooth muscle cells of the arterial wall in response to balloon injury. Both protein kinase C (PKC) and tyrosine kinase pp60(**src**) mediate augmented VEGF expression. In contrast, nitric oxide (NO) donors inhibit PKC-induced VEGF upregulation by interfering with binding of the transcription factor activator protein-1 (AP-1) to the VEGF promoter. Inhibition of VEGF promoter activation suggests that NO secreted by a restored endothelium functions as the negative feedback mechanism that downregulates VEGF expression to basal levels. Administration of a neutralizing VEGF antibody impaired reendothelialization following balloon injury performed in vivo. These findings establish a reciprocal relation between VEGF and NO in the endogenous regulation of endothelial integrity following arterial injury.

L121 ANSWER 52 OF 53 MEDLINE DUPLICATE 35

AB Vascular endothelial growth factor (VEGF) is a homodimeric peptide growth factor which binds to two structurally related tyrosine kinase receptors denoted Flt1 and KDR. In order to compare the signal transduction via these two receptors, the human Flt1 and KDR proteins were stably expressed in porcine aortic endothelial cells. Binding analyses using 125 I-VEGF revealed K_d values of 16 pM for Flt1 and 760 pM for KDR. Cultured human umbilical vein endothelial (HUVE) cells were found to express two distinct populations of binding sites with affinities similar to those for Flt1 and KDR, respectively. The KDR expressing cells showed striking changes in cell morphology, actin reorganization and membrane ruffling, chemotaxis and mitogenicity upon VEGF stimulation, whereas Flt1 expressing cells lacked such responses. KDR was found to undergo ligand-induced autophosphorylation in intact cells, and both Flt1 and KDR were phosphorylated in vitro in response to VEGF, however, KDR much more efficiently than Flt1. Neither the receptor-associated activity of phosphatidylinositol 3'-kinase nor tyrosine phosphorylation of phospholipase C- γ were affected by stimulation of Flt1 or KDR expressing cells, and phosphorylation of GTPase activating protein was

only slightly increased. Members of the **Src** family such as Fyn and **Yes** showed an increased level of phosphorylation upon **VEGF** stimulation of cells expressing Flt1 but not in cells expressing KDR. The maximal responses in KDR expressing porcine aortic endothelial cells were obtained at higher VEGF concentrations as compared to HUVE cells, i.e. in the presence of Flt1. This difference could possibly be explained by the formation of heterodimeric complexes between KDR and Flt1, or other molecules, in HUVE cells.

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